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YOU HAVE REQUESTED DATA FROM 23 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 23 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 2001:30133 CAPLUS  
DOCUMENT NUMBER: 134:120200  
TITLE: Role of the **DmpR**-mediated regulatory circuit  
in bacterial biodegradation properties in  
methylphenol-amended soils  
AUTHOR(S): Sarand, Inga; Skarfstad, Eleonore; Forsman, Mats;  
Romantschuk, Martin; Shingler, Victoria  
CORPORATE SOURCE: Department of Cell and Molecular Biology, Umea  
University, Umea, S-901 87, Swed.  
SOURCE: Appl. Environ. Microbiol. (2001), 67(1), 162-171  
CODEN: AEMIDF; ISSN: 0099-2240  
PUBLISHER: American Society for Microbiology  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB Pathway substrates and some structural analogs directly activate the  
regulatory protein **DmpR** to promote transcription of the *dmp*  
operon genes encoding the (methyl)phenol degradative pathway of  
*Pseudomonas* sp. strain CF600. While a wide range of phenols can activate  
**DmpR**, the location and nature of substituents on the basic  
phenolic ring can limit the level of activation and thus utilization of  
some compds. as assessed by growth on plates. We address the role of the  
arom. effector response of **DmpR** in detg. degradative properties  
in two soil matrixes that provide different nutritional conditions. Using  
the wild-type system and an isogenic counterpart contg. a **DmpR**  
**mutant** with enhanced ability to respond to para-substituted  
phenols, we demonstrate (1) that the enhanced in vitro biodegradative  
capacity of the regulator **mutant** strain is manifested in the two  
different soil types and (2) that exposure of the wild-type strain to  
4-methylphenol-contaminated soil led to rapid selection of a subpopulation  
exhibiting enhanced capacities to degrade the compd. Genetic and  
functional analyses of 10 of these derivs. demonstrated that all harbored  
a single **mutation** in the sensory domain of **DmpR** that  
mediated the phenotype in each case. These findings establish a  
dominating role for the arom. effector response of **DmpR** in detg.  
degrdn. properties. Results indicate that the ability to rapidly adapt  
regulator properties to different profiles of polluting compds. may  
underlie the evolutionary success of **DmpR**-like regulators in  
controlling arom. catabolic pathways.  
REFERENCE COUNT: 53  
REFERENCE(S):  
(1) Abril, M; J Bacteriol 1989, V171, P6782 CAPLUS  
(2) Ahn, Y; Biodegradation 1999, V10, P149 CAPLUS  
(3) Andersen, J; Appl Environ Microbiol 1998, V64,  
P2240 CAPLUS  
(4) Arai, H; Microbiology 1998, V144, P2895 CAPLUS  
(5) Ayoubi, P; Appl Environ Microbiol 1998, V64, P4353  
CAPLUS  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 2 OF 23 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 2000:43052 CAPLUS  
DOCUMENT NUMBER: 132:177993  
TITLE: Generation of novel bacterial regulatory proteins that  
detect priority pollutant phenols  
AUTHOR(S): Wise, Arlene A.; Kuske, Cheryl R.  
CORPORATE SOURCE: Environmental Molecular Biology Group, Los Alamos  
National Laboratory, Los Alamos, NM, 87545, USA  
SOURCE: Appl. Environ. Microbiol. (2000), 66(1), 163-169  
CODEN: AEMIDF; ISSN: 0099-2240  
PUBLISHER: American Society for Microbiology  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The genetic systems of bacteria that have the ability to use org.  
pollutants as carbon and energy sources can be adapted to create bacterial  
biosensors for the detection of industrial pollution. The creation of  
bacterial biosensors is hampered by a lack of information about the  
genetic systems that control prodn. of bacterial enzymes that metabolize

pollutants. We have attempted to overcome this problem through modification of **DmpR**, a regulatory protein for the **phenol** degrdn. pathway of *Pseudomonas* sp. strain CF600. The **phenol** detection capacity of **DmpR** was altered by using **mutagenic** PCR targeted to the **DmpR** sensor domain.

**DmpR mutants** were identified that both increased sensitivity to the phenolic effectors of wild-type **DmpR** and increased the range of mols. detected. The **phenol** detection characteristics of seven **DmpR mutants** were demonstrated through their ability to activate transcription of a lacZ reporter gene. Effectors of the **DmpR** derivs. included **phenol**, 2-chlorophenol, 2,4-dichlorophenol, 4-chloro-3-methylphenol, 2,4-dimethylphenol, 2-nitrophenol, and 4-nitrophenol.

REFERENCE COUNT: 41

REFERENCE(S):

- (3) Byrne, A; J Bacteriol 1996, V178, P6327 CAPLUS
  - (6) Delgado, A; J Biol Chem 1994, V269, P8059 CAPLUS
  - (7) Dower, W; Nucleic Acids Res 1988, V16, P6127 CAPLUS
  - (10) Heitzer, A; Appl Environ Microbiol 1992, V58, P1839 CAPLUS
  - (11) Ikariyama, Y; Anal Chem 1997, V69, P2600 CAPLUS
- ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 23 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1999:729100 CAPLUS

DOCUMENT NUMBER: 132:89115

TITLE:

Novel effector control through modulation of a preexisting binding site of the aromatic-responsive .sigma.54-dependent regulator **DmpR**

AUTHOR(S): O'Neill, Eric; Sze, Chun Chau; Shingler, Victoria

CORPORATE SOURCE: Department of Cell and Molecular Biology, Umea

University, Umea, S-901 87, Swed.

SOURCE: J. Biol. Chem. (1999), 274(45), 32425-32432

CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The *Pseudomonas* derived .sigma.54-dependent **DmpR** activator regulates transcription of the (methyl)**phenol** catabolic dmp-operon. **DmpR** is constitutively expressed, but its transcriptional promoting activity is pos. controlled in direct response to the presence of multiple arom. effectors. Previous work has led to a model in which effector binding by the amino-terminal region of the protein relieves repression of an intrinsic ATPase activity essential for its transcriptional promoting property. Here, the authors address whether the obsd. differences in the potencies of the multiple effectors (i) reside at the level of different arom. binding sites, or (ii) are mediated through differential binding affinities; furthermore, the authors address whether binding of distinct arom. effectors has different functional consequences for **DmpR** activity. These questions were addressed by comparing wild type and an effector specificity **mutant** of **DmpR** with respect to effector binding characteristics and the ability of aroms. to elicit ATPase activity and transcription. The results demonstrate that six test aroms. all share a common binding site on **DmpR** and that binding affinities det. the concn. at which **DmpR** responds to the presence of the effector, but not the magnitude of the responses. Interestingly, this anal. reveals that the novel abilities of the effector specificity **mutant** are not primarily due to acquisition of new binding abilities, but rather, they reside in being able to productively couple ATPase activity to transcriptional activation. The mechanistic implications of these findings in terms of arom. control of **DmpR** activity are discussed.

REFERENCE COUNT: 39

REFERENCE(S):

- (1) Abril, M; J Bacteriol 1989, V171, P6782 CAPLUS
  - (2) Austin, S; EMBO J 1992, V11, P2219 CAPLUS
  - (3) Austin, S; J Biol Chem 1994, V269, P18141 CAPLUS
  - (4) Berger, D; Proc Natl Acad Sci U S A 1994, V91, P103 CAPLUS
  - (5) Byrne, A; J Bacteriol 1996, V178, P6327 CAPLUS
- ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 4 OF 23 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 1997:391097 CAPLUS

DOCUMENT NUMBER: 127:105009

TITLE:

Studies on spontaneous promoter-up **mutations** in the transcriptional activator-encoding gene phlR and their effects on the degradation of **phenol** in *Escherichia coli* and *Pseudomonas putida*

AUTHOR(S): Burchhardt, G.; Schmidt, I.; Cuypers, H.; Petruschka, L.; Volker, A.; Herrmann, H.  
CORPORATE SOURCE: Institut Genetik Biochemie, Ernst-Moritz-Arndt Universitat Greifswald, Greifswald, D-17487, Germany  
SOURCE: Mol. Gen. Genet. (1997), 254(5), 539-547  
CODEN: MGGEAE; ISSN: 0026-8925  
PUBLISHER: Springer  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The activator-encoding gene phlR was identified upstream of the plasmid-encoded operon for **phenol** degrdn. in Pseudomonas putida strain H by cassette **mutagenesis** and DNA sequence anal. The deduced amino acid sequence of PHLR shows high homol. to **DmpR** of P. putida sp. CF600 and to the chromosomally encoded phhR of P. putida P35X reported previously. Trans-activation of **phenol** degrdn. was obsd. when phlR was overexpressed in a phlR insertion **mutant**. Transconjugants of Escherichia coli carrying pPGH11, which contains the complete set of phl genes, are unable to grow on **phenol** as carbon source. However, two types of **mutants** were selected for further characterization that were able to metabolize **phenol** as sole source of carbon and energy. In both types of **mutants** enhanced expression of phlR is responsible for the Phl+ phenotype. In type I (pPGH13) a deletion of 1 bp made the -35 region and the spacing between the -35 and -10 regions of the phlR promoter more similar to the consensus structure. In type II (pPGH14) a duplication of the phlR 5' region was identified that includes part of the -35 motif and reduces the spacing between the -35 and -10 regions. In addn., due to the duplication of part of phlR, the distance from the phlR promoter to the catabolic phl operon is increased. Different transcriptional start sites have been identified by primer extension anal. in clones harboring pPGH14 or the wild type phlR. Quant. primer extension anal. revealed that the greatest amt. of phlR transcript is expressed from the partial, phlR duplication. Growth on **phenol** and **phenol** hydroxylase activity reflect the high level of phlR transcript in E. coli transconjugants. Overexpression of PhlR was also obsd. when pPGH14 was transferred into P. putida, and results in earlier induction of the **phenol** degrdn. operon relative to the wild-type strain.

L3 ANSWER 5 OF 23 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 1997:117956 CAPLUS  
DOCUMENT NUMBER: 126:222650  
TITLE: Expression, inducer spectrum, domain structure, and function of MopR, the regulator of **phenol** degradation in Acinetobacter calcoaceticus NCIB8250  
AUTHOR(S): Schirmer, Falck; Ehrt, Sabine; Hillen, Wolfgang  
CORPORATE SOURCE: Institut fuer Mikrobiologie, Biochemie und Genetik der Friedrich-Alexander, Universitaet Erlangen-Nuernberg, Erlangen, 91058, Germany  
SOURCE: J. Bacteriol. (1997), 179(4), 1329-1336  
CODEN: JOBAAY; ISSN: 0021-9193  
PUBLISHER: American Society for Microbiology  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB Degrdn. of PhOH by A. calcoaceticus NCIB8250 involves .sigma.54-dependent expression of a multicomponent PhOH hydroxylase and catechol 1,2-dioxygenase encoded by the mop operon. Complementation of a new **mutant** deficient in PhOH utilization yielded the regulatory locus **mopR**. It is located in divergent orientation next to the **mop** operon. MopR is constitutively expressed at a low level from a .sigma.70-type promoter and belongs to the NtrC family of regulators. The amino acid sequence is similar to that of **XylR** regulating xylene degrdn. and to that of **DmpR** regulating dimethylphenol degrdn. in Pseudomonas spp. However, it shows a different effector profile for substituted phenols than **DmpR**. MopR activates **phenol** hydroxylase expression in the presence of PhOH in Escherichia coli, indicating that it binds the effector. The PhOH-binding A domains of MopR and **DmpR** have fewer identical residues than the A domains of **DmpR** and **XylR**, despite the fact that **XylR** recognizes different effectors. This suggests that sequence conservation in the A domain does not reflect the potential to bind the resp. effectors. Overexpression of the MopR A domain in the presence of wild-type MopR causes loss of **mop** inducibility by PhOH, establishing its neg. transdominance over MopR. Deletion of 110 residues from the N terminus did not affect transdominance of the truncated domain, whereas deletion of 150 residues abolished it completely. This result established the distinction of 2 subdomains, AN and AC, which together constitute the A domain. The C-terminal portion of the A domain, AC, shows considerable affinity for the C domain, even in the presence of the trigger PhOH.

ACCESSION NUMBER: 1996:452557 CAPLUS  
DOCUMENT NUMBER: 125:108278  
TITLE: Genetic evidence for interdomain regulation of the phenol-responsive .sigma.54-dependent activator DmpR  
AUTHOR(S): Ng, Lee Ching; O'Neill, Eric; Shingler, Victoria  
CORPORATE SOURCE: Dep. Cell Molecular Biology, Umea Univ., Umea, S-901 87, Swed.  
SOURCE: J. Biol. Chem. (1996), 271(29), 17281-17286  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The .sigma.54-dependent **DmpR** activator regulates transcription of the dmp operon that encodes the enzymes for catabolism of (methyl)phenols. **DmpR** is expressed constitutively, but its transcriptional promoting activity is controlled pos. in direct response to the presence of arom. pathway substrates (effectors). **DmpR** has a distinct domain structure with the amino-terminal A-domain controlling the specificity of activation of the regulator by arom. effectors (signal reception), a central C-domain mediating an ATPase activity essential for transcription activation, and a carboxyl-terminal D-domain involved in DNA binding. Deletion of the A-domain has been shown previously to result in an effector-independent transcriptional activator with constitutive ATPase activity. These results, in conjunction with the location of **mutations** within the A- and C-domains which exhibit an effector-independent (semiconstitutive) property, have led to a working model in which the A-domain serves to mask the ATPase and transcriptional promoting activity of the C-domain in the absence of effectors. To investigate the mechanism by which the A-domain exerts its repressive effect, we developed a genetic system to select pos. for intramol. second site revertants of **DmpR**. The results demonstrate (i) that **mutations** within the A-domain can suppress the semiconstitutive activity of C-domain located **mutations** and vice versa; (ii) that the C-domain located **mutations** do not influence the intrinsic ATPase and transcriptional promoting property of the C-domain in the absence of the A-domain; and (iii) that semiconstitutive **mutations** of the A- and C-domain have an additive effect. Taken together these results support a model in which the A-domain represses the function(s) of the C-domain by direct interactions between residues of the two domains.

L3 ANSWER 7 OF 23 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 1995:817447 CAPLUS  
DOCUMENT NUMBER: 123:221557  
TITLE: Direct regulation of the ATPase activity of the transcriptional activator **DmpR** by aromatic compounds  
AUTHOR(S): Shingler, V.; Pavel H.  
CORPORATE SOURCE: Department of Cell and Molecular Biology, Umeae University, Umeae, S-901 87, Swed.  
SOURCE: Mol. Microbiol. (1995), 17(3), 505-13  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB The NtrC-like regulator **DmpR** controls transcription from the dmp operon that encodes the enzymes for catabolism of phenol and some related arom. compds. **DmpR** activates transcription from the .sigma.54-dependent dmp-operon promoter in the presence of pathway substrates or structural analogs in the growth medium. Using affinity-purified **DmpR** and a truncated deriv., we show here that arom. compds. directly activate the ATPase activity of this protein in vitro, and that the amino-terminal domain represses this activity in the absence of an arom. ligand. In order to dissect the activation process, derivs. of **DmpR** exhibiting single amino acid changes were isolated and their effector-dependence and specificity profiles were analyzed in vivo. The mechanistic implications of the phenotypes of these **mutants** are discussed.

L3 ANSWER 8 OF 23 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 1993:422129 CAPLUS  
DOCUMENT NUMBER: 119:22129  
TITLE: Cloning and nucleotide sequence of the gene encoding the positive regulator (**DmpR**) of the phenol catabolic pathway encoded by pVI150 and identification of **DmpR** as a member of the NtrC family of transcriptional activators  
AUTHOR(S): Shingler, Victoria; Bartilson, Magdalena; Moore, Terry  
CORPORATE SOURCE: Dep. Cell Mol. Biol., Univ. Umea, Umea, S-901 87, Swed.  
SOURCE: J. Bacteriol. (1993), 175(6), 1596-604  
CODEN: JOBAAY; ISSN: 0021-9193

DOCUMENT TYPE: Journal  
LANGUAGE: English

AB The catabolic plasmid pVI150 of *Pseudomonas* sp. strain CF600 encodes all the genetic information required for the regulated metab. of **phenol** and some of its methyl-substituted derivs. The structural dmp genes of the pathway are clustered in a single operon that lies just downstream of a -24 TGGC, -12 TTGC nif/ntr-like promoter sequence. Promoters of this class are recognized by a minor form of RNA polymerase utilizing .sigma.54 (NtrA, RpoN). Primer extension anal. demonstrated that the dmp operon transcript initiates downstream of the -24, -12 promoter. Transposon insertion **mutants**, specifically defective in the regulation of the dmp operon, were isolated, and complementation of a **phenol**-utilization regulatory **mutant** was used to identify the regulatory locus, **dmpR**. The 67-kDa **dmpR** gene product alone was shown to be sufficient for activation of transcription from the dmp operon promoter. Nucleotide sequence detn. revealed that **DmpR** belongs to the NtrC family of transcriptional activators that regulate transcription from -24, -12 promoters. The deduced amino acid sequence of **DmpR** has high homol. (40 to 67% identity) with the central and carboxy-terminal regions of these activators, which are believed to be involved in the interaction with the .sigma.54 RNA polymerase and in DNA binding, resp. The amino-terminal region of **DmpR** was found to share 64% identity with the amino-terminal region of **XylR**, which is also a member of this family of activators. This region has been implicated in effector recognition of arom. compds. that is required for the regulatory activity of **XylR**.

L3 ANSWER 9 OF 23 USPATFULL

ACCESSION NUMBER: 2001:10723 USPATFULL  
TITLE: Method to isolate **mutants** and to clone the complementing gene  
INVENTOR(S): De Graaff, Leendert Hendrik, Oosterbeek, Netherlands  
Van Den Broeck, Henrietta Catharina, Bennekom, Netherlands  
Visser, Jacob, Wageningen, Netherlands  
PATENT ASSIGNEE(S): Danisco Ingredients A/S (Danisco A/S), Brabrand, Denmark (non-U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6177261	20010123
	WO 9700962	19970109
APPLICATION INFO.:	US 1997-981729	19971223 (8)
	WO 1996-NL259	19960624 19971223 PCT 371 date 19971223 PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	EP 1995-20107	19950623
	EP 1995-202346	19950830
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Schwartzman, Robert A.	
LEGAL REPRESENTATIVE:	Sughrue, Mion, Zinn, Macpeak & Seas, PLLC	
NUMBER OF CLAIMS:	32	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	3 Drawing Figure(s); 3 Drawing Page(s)	
LINE COUNT:	2466	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The subject invention lies in the field of microorganism **mutation** and selection of the **mutants**. In particular, the invention is directed at obtaining metabolic **mutants** in a simple, direct and specific manner. In a preferred embodiment it is also possible to obtain desired **mutants** not comprising recombinant DNA, thereby facilitating incorporation thereof in products for human consumption or application, due to shorter legislative procedures. The method according to the invention involves random **mutation** and specific selection of the desired metabolic **mutant**. A nucleic acid cassette comprising a nucleic acid sequence encoding a bidirectional marker, said nucleic acid cassette further comprising a basic transcriptional unit operatively linked to the nucleic acid sequence encoding the bidirectional marker and said nucleic acid cassette further comprising an inducible enhancer or activator sequence linked to the basic transcription unit in such a manner that upon induction of the enhancer or activator sequence the bidirectional marker encoding nucleic acid sequence is expressed, said inducible enhancer or activator sequence being driven from a gene associated with metabolism is claimed as is application thereof in a selection method for **mutants**. In addition a regular gene xlnR encoding an activating

regulator of an inducible enhancer or activator sequence and application of said gene and/or its expression product in overexpression of homologous or heterologous protein or peptide is described. Knockout **mutants** wherein said gene is absent or inactivated and **mutants** with increased or decreased DNA binding capacity are also claimed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 10 OF 23 USPATFULL  
ACCESSION NUMBER: 2000:84053 USPATFULL  
TITLE: Biotechnological method of producing biotin  
INVENTOR(S): Birch, Olwen, Naters, Switzerland  
Brass, Johann, Ausserberg, Switzerland  
Fuhrmann, Martin, Visp, Switzerland  
Shaw, Nicholas, Visp, Switzerland  
PATENT ASSIGNEE(S): Lonza A.G., Basel, Switzerland (non-U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 6083712	20000704
	WO 9408023	19940414
APPLICATION INFO.:	US 1995-411768	19950608 (8)
	WO 1993-EP2688	19931001 19950608 PCT 371 date 19950608 PCT 102(e) date

	NUMBER	DATE
PRIORITY INFORMATION:	CH 1992-3124	19921002
	CH 1993-2134	19930715
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Carlson, Karen Cochrane	
LEGAL REPRESENTATIVE:	Baker & Botts, L.L.P.	
NUMBER OF CLAIMS:	30	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	16 Drawing Figure(s); 16 Drawing Page(s)	
LINE COUNT:	2589	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB In DNA fragments and plasmids comprising the bioB, bioF, bioC, bioD and bioA genes responsible for biosynthesis of biotin, or their functionally equivalent genetic variants and **mutants** from enteric bacteria, the genes are arranged in a transcription unit. These DNA fragments and plasmids can be contained in microorganisms which can be used to produce biotin.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 11 OF 23 USPATFULL  
ACCESSION NUMBER: 91:60746 USPATFULL  
TITLE: Expression system with trans-acting DNA segments  
INVENTOR(S): Hastrup, Sven, Copenhagen, Denmark  
PATENT ASSIGNEE(S): Novo-Nordisk A/S, Denmark (non-U.S. corporation)

	NUMBER	DATE
PATENT INFORMATION:	US 5036002	19910730
APPLICATION INFO.:	US 1987-39298	19870417 (7)

	NUMBER	DATE
PRIORITY INFORMATION:	DK 1986-1777	19860417
DOCUMENT TYPE:	Utility	
PRIMARY EXAMINER:	Teskin, Robin	
ASSISTANT EXAMINER:	Ellis, Joan	
LEGAL REPRESENTATIVE:	Sterne, Kessler, Goldstein & Fox	
NUMBER OF CLAIMS:	13	
EXEMPLARY CLAIM:	1	
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 9 Drawing Page(s)	
LINE COUNT:	505	

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Gene expression systems comprising an expression vector and a "trans-acting DNA segment", where the expression vector comprises the gene or genes to be expressed and one or more cis-acting regulatory elements which are responsive to a trans-acting factor produced by said "trans-acting DNA segment". More specifically the invention relates to such gene expression systems where said "trans-acting DNA segment" and said cis-acting regulatory elements comprise one or more segments of the genome from a *Bacillus* species. Methods for stimulating the production of gene products, vectors for transforming microorganisms, and their use

are also disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 12 OF 23 EUROPATFULL COPYRIGHT 2001 WILA

PATENT APPLICATION - PATENTANMELDUNG - DEMANDE DE BREVET

ACCESSION NUMBER: 1059354 EUROPATFULL EW 200050 FS OS  
TITLE: Sequence-determined DNA fragments and corresponding polypeptides encoded thereby.  
DNS-fragmente mit bestimmter Sequenz und die dadurch kodierte Polypeptide.  
Fragments d'ADN avec des sequences determinees et polypeptides encodees par lesdits fragments.  
INVENTOR(S): Alexandrov, Nickolai, 1404 Oak Trail St., Thousand Oaks, CA 91320, US;  
Troukhan, Maxim E., 1675 Amberwood Dr. No. 2, South Pasadena, CA 91030, US  
Ceres Incorporated, 3007 Malibu Canyon Road, Malibu, CA 90265, US  
PATENT ASSIGNEE(S): 2967260  
PATENT ASSIGNEE NO:  
AGENT: Bannerman, David Gardner et al., Withers & Rogers,  
Goldings House, 2 Hays Lane, London SE1 2HW, GB  
AGENT NUMBER: 28001  
OTHER SOURCE: BEPA2000096 EP 1059354 A2 0418  
SOURCE: Wila-EPZ-2000-H50-T1a  
DOCUMENT TYPE: Patent  
LANGUAGE: Anmeldung in Englisch; Veroeffentlichung in Englisch  
DESIGNATED STATES: R AT; R BE; R CH; R CY; R DE; R DK; R ES; R FI; R FR; R GB; R GR; R IE; R IT; R LI; R LU; R MC; R NL; R PT; R SE; R AL; R LT; R LV; R MK; R RO; R SI  
PATENT INFO.PUB.TYPE: EPA2 EUROPAEISCHE PATENTANMELDUNG  
PATENT INFORMATION:

PATENT NO	KIND DATE
EP 1059354	A2 20001213
	20001213
APPLICATION INFO.: EP 2000-304943	20000612
PRIORITY APPLN. INFO.: US 1999-138540	19990610
US 1999-138847	19990610

L3 ANSWER 13 OF 23 EUROPATFULL COPYRIGHT 2001 WILA

PATENT APPLICATION - PATENTANMELDUNG - DEMANDE DE BREVET

ACCESSION NUMBER: 1033405 EUROPATFULL EW 200036 FS OS  
TITLE: Sequence-determined DNA fragments and corresponding polypeptides encoded thereby.  
DNS-fragmente mit bestimmter Sequenz und die dadurch kodierte Polypeptide.  
Fragments d'ADN avec des sequences determinees et polypeptides encodees par lesdits fragments.  
INVENTOR(S): Alexandrov, Nickolai, 1404 Oak Trail St., Thousand Oaks, CA 91320, US;  
Brover, Vyacheslav, 5916 N. Las Virgenes Rd. #590, Calabasas, CA 91302, US;  
Chen, Xianfeng, 1705 S. Westgate Ave. #2, Los Angeles, CA 90025, US;  
Subramanian, Gopalakrishnan, 4205 Peach Slope Rd., Moorpark, CA 93021, US;  
Troukhan, Maxim E., 1675 Amberwood Dr. #2, South Pasadena, CA 91030, US;  
Zheng, Liansheng, 12333 Wild Turkey Court, #B, Creve Coeur, MO 63141, US;  
Dumas, J., US  
PATENT ASSIGNEE(S): Ceres Incorporated, 3007 Malibu Canyon Road, Malibu, CA 90265, US  
PATENT ASSIGNEE NO:  
AGENT: 2967260  
AGENT NUMBER: Bannerman, David Gardner et al., Withers & Rogers,  
Goldings House, 2 Hays Lane, London SE1 2HW, GB  
AGENT NUMBER: 28001  
OTHER SOURCE: BEPA2000068 EP 1033405 A2 0344  
SOURCE: Wila-EPZ-2000-H36-T1a  
DOCUMENT TYPE: Patent  
LANGUAGE: Anmeldung in Englisch; Veroeffentlichung in Englisch  
DESIGNATED STATES: R AT; R BE; R CH; R CY; R DE; R DK; R ES; R FI; R FR; R GB; R GR; R IE; R IT; R LI; R LU; R MC; R NL; R PT; R SE; R AL; R LT; R LV; R MK; R RO; R SI  
PATENT INFO.PUB.TYPE: EPA2 EUROPAEISCHE PATENTANMELDUNG

## PATENT INFORMATION:

	PATENT NO	KIND DATE
'OFFENLEGUNGS' DATE:	EP 1033405	A2 20000906
APPLICATION INFO.:	EP 2000-301439	20000906
PRIORITY APPLN. INFO.:	US 1999-121825	20000225
	US 1999-123180	19990225
	US 1999-123548	19990305
	US 1999-125788	19990309
	US 1999-126264	19990323
	US 1999-126785	19990325
	US 1999-127462	19990329
	US 1999-128234	19990401
	US 1999-128714	19990406
	US 1999-129845	19990416
	US 1999-130077	19990419
	US 1999-130449	19990421
	US 1999-130891	19990423
	US 1999-130510	19990423
	US 1999-131449	19990428
	US 1999-132407	19990430
	US 1999-132048	19990430
	US 1999-132484	19990504
	US 1999-132485	19990505
	US 1999-132487	19990506
	US 1999-132486	19990506
	US 1999-132863	19990507
	US 2000-176866	20000119
	US 2000-176867	20000119
	US 2000-176910	20000119
	US 2000-178166	20000126
	US 2000-178545	20000127
	US 2000-178547	20000127
	US 2000-177666	20000127
	US 2000-178546	20000127
	US 2000-178544	20000127
	US 2000-178754	20000128
	US 2000-178755	20000128
	US 2000-179388	20000201
	US 2000-179395	20000201
	US 2000-180139	20000203
	US 2000-180039	20000203
	US 2000-180206	20000204
	US 2000-180207	20000204
	US 2000-180696	20000207
	US 2000-180695	20000207
	US 2000-181214	20000209
	US 2000-181228	20000209
	US 2000-181551	20000210
	US 2000-181476	20000210
	US 2000-182478	20000215
	US 2000-182477	20000215
	US 2000-182516	20000215
	US 2000-182512	20000215
	US 2000-183166	20000217
	US 2000-183165	20000217

L3 ANSWER 14 OF 23 EUROPATFULL COPYRIGHT 2001 WILA

PATENT APPLICATION - PATENTANMELDUNG - DEMANDE DE BREVET

ACCESSION NUMBER: 798384 EUROPATFULL EW 199740 FS OS  
 TITLE: Biotechnological method of producing biotin.  
                  Biotechnologisches Verfahren zur Herstellung von Biotin.  
                  Procede biotechnologique de preparation de biotine.

INVENTOR(S): Birch, Olwen, Dammweg 11D, 3904 Naters, CH;  
                  Brass, Johann, In den Schatmatten, 3938 Ausserberg, CH;  
                  Fuhrmann, Martin, Am Balkenweg 23, 4460 Gelterkinden,  
                  CH;

PATENT ASSIGNEE(S): Shaw, Nicholas, Weingartenweg 14, 3930 Visp, CH  
 LONZA A.G., CH-3945 Gampel/Wallis, CH

PATENT ASSIGNEE NO: 425663

AGENT: KUHNEN, WACKER & PARTNER, Alois-Steinecker-Strasse 22,  
 85354 Freising, DE

AGENT NUMBER: 100053

OTHER SOURCE: ESP1997060 EP 0798384 A1 971001

SOURCE: Wila-EPZ-1997-H40-T1a

DOCUMENT TYPE: Patent

LANGUAGE: Anmeldung in Deutsch; Veröffentlichung in Deutsch

DESIGNATED STATES: R AT; R BE; R CH; R DE; R DK; R ES; R FR; R GB; R IE; R IT; R LI; R NL; R PT; R SE

PATENT INFO.PUB.TYPE: EPA1 EUROPÄISCHE PATENTANMELDUNG

PATENT INFORMATION:

	PATENT NO	KIND	DATE
'OFFENLEGUNGS' DATE:	EP 798384	A1	19971001
APPLICATION INFO.:	EP 1997-107803		19971001
PRIORITY APPLN. INFO.:	CH 1992-3124		19921002
	CH 1993-2134		19930715
RELATED DOC. INFO.:	EP 667909	DIV	

L3 ANSWER 15 OF 23 MEDLINE

ACCESSION NUMBER: 2000270126 MEDLINE

DOCUMENT NUMBER: 20270126

TITLE: Identification of an effector specificity subregion within the aromatic-responsive regulators **DmpR** and **XylR** by DNA shuffling.

AUTHOR: Skarfstad E; O'Neill E; Garmendia J; Shingler V

CORPORATE SOURCE: Department of Cell and Molecular Biology, Umeå University, Umeå, Sweden.

SOURCE: JOURNAL OF BACTERIOLOGY, (2000 Jun) 182 (11) 3008-16.  
Journal code: HH3. ISSN: 0021-9193.

PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200008

ENTRY WEEK: 20000802

AB The *Pseudomonas* derived sigma(54)-dependent regulators **DmpR** and **XylR** control the expression of genes involved in catabolism of aromatic compounds. Binding to distinct, nonoverlapping groups of aromatic effectors controls the activities of these transcriptional activators. Previous work has derived a common mechanistic model for these two regulators in which effector binding by the N-terminal 210 residues (the A-domain) of the protein relieves repression of an intrinsic ATPase activity essential for its transcription-promoting property and allows productive interaction with the transcriptional apparatus. Here we dissect the A-domains of **DmpR** and **XylR** by DNA shuffling to identify the region(s) that mediates the differences in the effector specificity profiles. Analysis of *in vivo* transcription in response to multiple aromatic effectors and the *in vitro* phenol-binding abilities of regulator derivatives with hybrid **DmpR/XylR** A-domains reveals that residues 110 to 186 are key determinants that distinguish the effector profiles of **DmpR** and **XylR**. Moreover, the properties of some mosaic **DmpR/XylR** derivatives reveal that high-affinity aromatic effector binding can be completely uncoupled from the ability to promote transcription. Hence, novel aromatic binding properties will only be translated into functional transcriptional activation if effector binding also triggers release of interdomain repression.

L3 ANSWER 16 OF 23 MEDLINE

ACCESSION NUMBER: 2000087532 MEDLINE

DOCUMENT NUMBER: 20087532

TITLE: Generation of novel bacterial regulatory proteins that detect priority pollutant phenols.

AUTHOR: Wise A A; Kuske C R

CORPORATE SOURCE: Environmental Molecular Biology Group, Biosciences Division, Los Alamos National Laboratory, Los Alamos, New Mexico 87545, USA.

SOURCE: APPLIED AND ENVIRONMENTAL MICROBIOLOGY, (2000 Jan) 66 (1) 163-9.

Journal code: 6K6. ISSN: 0099-2240.

PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200004

ENTRY WEEK: 20000402

AB The genetic systems of bacteria that have the ability to use organic pollutants as carbon and energy sources can be adapted to create bacterial biosensors for the detection of industrial pollution. The creation of bacterial biosensors is hampered by a lack of information about the genetic systems that control production of bacterial enzymes that metabolize pollutants. We have attempted to overcome this problem through modification of **DmpR**, a regulatory protein for the phenol degradation pathway of *Pseudomonas* sp. strain CF600. The phenol detection capacity of **DmpR** was altered by using

**mutagenic** PCR targeted to the **DmpR** sensor domain. **DmpR** mutants were identified that both increased sensitivity to the phenolic effectors of wild-type **DmpR** and increased the range of molecules detected. The **phenol** detection characteristics of seven **DmpR** mutants were demonstrated through their ability to activate transcription of a lacZ reporter gene. Effectors of the **DmpR** derivatives included **phenol**, 2-chlorophenol, 2,4-dichlorophenol, 4-chloro-3-methylphenol, 2,4-dimethylphenol, 2-nitrophenol, and 4-nitrophenol.

L3 ANSWER 17 OF 23 MEDLINE  
ACCESSION NUMBER: 2000011451 MEDLINE  
DOCUMENT NUMBER: 20011451  
TITLE: Novel effector control through modulation of a preexisting binding site of the aromatic-responsive sigma(54)-dependent regulator **DmpR**.  
AUTHOR: O'Neill E; Sze C C; Shingler V  
CORPORATE SOURCE: Department of Cell and Molecular Biology, Umea University, S-901 87 Umea, Sweden.  
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Nov 5) 274 (45) 32425-32.  
PUB. COUNTRY: United States  
LANGUAGE: English  
FILE SEGMENT: Priority Journals; Cancer Journals  
ENTRY MONTH: 200002  
ENTRY WEEK: 20000204  
AB The Pseudomonas derived sigma(54)-dependent **DmpR** activator regulates transcription of the (methyl)**phenol** catabolic dmp-operon. **DmpR** is constitutively expressed, but its transcriptional promoting activity is positively controlled in direct response to the presence of multiple aromatic effectors. Previous work has led to a model in which effector binding by the amino-terminal region of the protein relieves repression of an intrinsic ATPase activity essential for its transcriptional promoting property. Here, we address whether the observed differences in the potencies of the multiple effectors (i) reside at the level of different aromatic binding sites, or (ii) are mediated through differential binding affinities; furthermore, we address whether binding of distinct aromatic effectors has different functional consequences for **DmpR** activity. These questions were addressed by comparing wild type and an effector specificity **mutant** of **DmpR** with respect to effector binding characteristics and the ability of aromatics to elicit ATPase activity and transcription. The results demonstrate that six test aromatics all share a common binding site on **DmpR** and that binding affinities determine the concentration at which **DmpR** responds to the presence of the effector, but not the magnitude of the responses. Interestingly, this analysis reveals that the novel abilities of the effector specificity **mutant** are not primarily due to acquisition of new binding abilities, but rather, they reside in being able to productively couple ATPase activity to transcriptional activation. The mechanistic implications of these findings in terms of aromatic control of **DmpR** activity are discussed.

L3 ANSWER 18 OF 23 MEDLINE  
ACCESSION NUMBER: 97340939 MEDLINE  
DOCUMENT NUMBER: 97340939  
TITLE: Studies on spontaneous promoter-up **mutations** in the transcriptional activator-encoding gene phIR and their effects on the degradation of **phenol** in *Escherichia coli* and *Pseudomonas putida*.  
AUTHOR: Burchhardt G; Schmidt I; Cuypers H; Petruschka L; Volker A; Herrmann H  
CORPORATE SOURCE: Institut fur Genetik und Biochemie, Ernst-Moritz-Arndt Universitat Greifswald, Germany.  
SOURCE: MOLECULAR AND GENERAL GENETICS, (1997 May 20) 254 (5) 539-47.  
PUB. COUNTRY: GERMANY: Germany, Federal Republic of  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
OTHER SOURCE: GENBANK-X91145  
ENTRY MONTH: 199709  
AB The activator-encoding gene phIR was identified upstream of the plasmid-encoded operon for **phenol** degradation in *Pseudomonas putida* strain H by cassette **mutagenesis** and DNA sequence analysis. The deduced amino acid sequence of PHLR shows high homology to **DmpR** of *P. putida* sp. CF600 and to the chromosomally encoded PhhR

of *P. putida* P35X reported previously. Trans-activation of **phenol** degradation was observed when *phlR* was overexpressed in a *phlR* insertion **mutant**. Transconjugants of *Escherichia coli* carrying pPGH11, which contains the complete set of *phl* genes, are unable to grow on **phenol** as carbon source. However, two types of **mutants** were selected for further characterization that were able to metabolize **phenol** as sole source of carbon and energy. In both types of **mutants** enhanced expression of *phlR* is responsible for the *Phl<sup>+</sup>* phenotype. In type I (pPGH13) a deletion of 1 bp made the -35 region and the spacing between the -35 and -10 regions of the *phlR* promoter more similar to the consensus structure. In type II (pPGH14) a duplication of the *phlR* 5' region was identified that includes part of the -35 motif and reduces the spacing between the -35 and -10 regions. In addition, due to the duplication of part of *phlR*, the distance from the *phlR* promoter to the catabolic *phl* operon is increased. Different transcriptional start sites have been identified by primer extension analysis in clones harboring pPGH14 or the wild type *phlR*. Quantitative primer extension analysis revealed that the greatest amount of *phlR* transcript is expressed from the partial, *phlR* duplication. Growth on **phenol** and **phenol** hydroxylase activity reflect the high level of *phlR* transcript in *E. coli* transconjugants. Overexpression of *PhlR* was also observed when pPGH14 was transferred into *P. putida*, and results in earlier induction of the **phenol** degradation operon relative to the wild-type strain.

L3 ANSWER 19 OF 23 MEDLINE

ACCESSION NUMBER: 97175564 MEDLINE

DOCUMENT NUMBER: 97175564

TITLE: Expression, inducer spectrum, domain structure, and function of *MopR*, the regulator of **phenol** degradation in *Acinetobacter calcoaceticus* NCIB8250.

AUTHOR: Schirmer F; Ehrt S; Hillen W

CORPORATE SOURCE: Lehrstuhl fur Mikrobiologie, Institut fur Mikrobiologie, Biochemie und Genetik der Friedrich-Alexander Universitat Erlangen-Nurnberg, Erlangen, Germany.

SOURCE: JOURNAL OF BACTERIOLOGY, (1997 Feb) 179 (4) 1329-36.

Journal code: HH3. ISSN: 0021-9193.

PUB. COUNTRY: United States

LANGUAGE: English

FILE SEGMENT: Priority Journals

OTHER SOURCE: GENBANK-Z69251

ENTRY MONTH: 199705

AB Degradation of **phenol** by *Acinetobacter calcoaceticus* NCIB8250 involves (*sigma*54-dependent expression of a multicomponent **phenol** hydroxylase and catechol 1,2-dioxygenase encoded by the *mop* operon. Complementation of a new **mutant** deficient in **phenol** utilization yielded the regulatory locus *mopR*. It is located in divergent orientation next to the *mop* operon. *MopR* is constitutively expressed at a low level from a *sigma*70-type promoter and belongs to the *NtrC* family of regulators. The amino acid sequence is similar to that of *XylR* regulating xylene degradation and to that of *DmpR* regulating dimethylphenol degradation in *Pseudomonas* spp. However, it shows a different effector profile for substituted phenols than *DmpR*. *MopR* activates **phenol** hydroxylase expression in the presence of **phenol** in *Escherichia coli*, indicating that it binds the effector. The **phenol** binding A domains of *MopR* and *DmpR* have fewer identical residues than the A domains of *DmpR* and *XylR*, despite the fact that *XylR* recognizes different effectors. This suggests that sequence conservation in the A domain does not reflect the potential to bind the respective effectors. Overexpression of the *MopR* A domain in the presence of wild-type *MopR* causes loss of *mop* inducibility by **phenol**, establishing its negative transdominance over *MopR*. Deletion of 110 residues from the N terminus did not affect transdominance of the truncated domain, whereas deletion of 150 residues abolished it completely. This result establishes the distinction of two subdomains, A(N) and A(C), which together constitute the A domain. The C-terminal portion of the A domain, A(C), shows considerable affinity for the C domain, even in the presence of the trigger **phenol**.

L3 ANSWER 20 OF 23 MEDLINE

ACCESSION NUMBER: 96291880 MEDLINE

DOCUMENT NUMBER: 96291880

TITLE: Genetic evidence for interdomain regulation of the **phenol**-responsive final *sigma*54-dependent activator *DmpR*.

AUTHOR: Ng L C; O'Neill E; Shingler V

CORPORATE SOURCE: Department of Cell and Molecular Biology, Umea University, S-901 87 Umea, Sweden.

SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 Jul 19) 271 (29)

17281-6.  
Journal code: HIV. ISSN: 0021-9258.  
PUB. COUNTRY: United States  
LANGUAGE: English  
FILE SEGMENT: Priority Journals; Cancer Journals  
ENTRY MONTH: 199611

AB The final sigma54-dependent **DmpR** activator regulates transcription of the dmp operon that encodes the enzymes for catabolism of (methyl)phenols. **DmpR** is expressed constitutively, but its transcriptional promoting activity is controlled positively in direct response to the presence of aromatic pathway substrates (effectors). **DmpR** has a distinct domain structure with the amino-terminal A-domain controlling the specificity of activation of the regulator by aromatic effectors (signal reception), a central C-domain mediating an ATPase activity essential for transcriptional activation, and a carboxyl-terminal D-domain involved in DNA binding. Deletion of the A-domain has been shown previously to result in an effector-independent transcriptional activator with constitutive ATPase activity. These results, in conjunction with the location of **mutations** within the A- and C-domains which exhibit an effector-independent (semiconstitutive) property, have led to a working model in which the A-domain serves to mask the ATPase and transcriptional promoting activity of the C-domain in the absence of effectors. To investigate the mechanism by which the A-domain exerts its repressive effect, we developed a genetic system to select positively for intramolecular second site revertants of **DmpR**. The results demonstrate (i) that **mutations** within the A-domain can suppress the semiconstitutive activity of C-domain located **mutations** and vice versa; (ii) that the C-domain located **mutations** do not influence the intrinsic ATPase and transcriptional promoting property of the C-domain in the absence of the A-domain; and (iii) that semiconstitutive **mutations** of the A- and C-domain have an additive effect. Taken together these results support a model in which the A-domain represses the function(s) of the C-domain by direct interactions between residues of the two domains.

L3 ANSWER 21 OF 23 MEDLINE  
ACCESSION NUMBER: 96100449 MEDLINE  
DOCUMENT NUMBER: 96100449  
TITLE: Direct regulation of the ATPase activity of the transcriptional activator **DmpR** by aromatic compounds.  
AUTHOR: Shingler V; Pavel H  
CORPORATE SOURCE: Department of Cell and Molecular Biology, Umea University, Sweden.  
SOURCE: MOLECULAR MICROBIOLOGY, (1995 Aug) 17 (3) 505-13.  
Journal code: MOM. ISSN: 0950-382X.  
PUB. COUNTRY: ENGLAND: United Kingdom  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199605

AB The NtrC-like regulator **DmpR** controls transcription from the dmp operon that encodes the enzymes for catabolism of **phenol** and some related aromatic compounds. **DmpR** activates transcription from the sigma 54-dependent dmp-operon promoter in the presence of pathway substrates or structural analogues in the growth medium. Using affinity-purified **DmpR** and a truncated derivative, we show here that aromatic compounds directly activate the ATPase activity of this protein *in vitro*, and that the amino-terminal domain represses this activity in the absence of an aromatic ligand. In order to dissect the activation process, derivatives of **DmpR** exhibiting single amino acid changes were isolated and their effector-dependence and specificity profiles were analysed *in vivo*. The mechanistic implications of the phenotypes of these **mutants** are discussed.

L3 ANSWER 22 OF 23 MEDLINE  
ACCESSION NUMBER: 95095924 MEDLINE  
DOCUMENT NUMBER: 95095924  
TITLE: An aromatic effector specificity **mutant** of the transcriptional regulator **DmpR** overcomes the growth constraints of *Pseudomonas* sp. strain CF600 on para-substituted methylphenols.  
AUTHOR: Pavel H; Forsman M; Shingler V  
CORPORATE SOURCE: Department of Cell and Molecular Biology, Umea University, Sweden.  
SOURCE: JOURNAL OF BACTERIOLOGY, (1994 Dec) 176 (24) 7550-7.  
Journal code: HH3. ISSN: 0021-9193.  
PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199503  
AB The pVI150 catabolic plasmid of *Pseudomonas* sp. strain CF600 carries the dmp system, which comprises the divergently transcribed **dmpR** gene and the dmp operon coding for the catabolic enzymes required for growth on (methyl)phenols. The constitutively expressed **DmpR** transcriptional activator positively controls the expression of the RpoN-dependent dmp operon promoter in the presence of the aromatic effector in the growth medium. However, the magnitude of the transcriptional response differs depending on the position of the methyl substituent on the aromatic ring. Experiments involving an elevated copy number of the dmp system demonstrate that growth on para-substituted methylphenols is limited by the level of the catabolic enzymes. An effector specificity **mutant** of **DmpR**, **DmpR**-E135K, that responded to the presence of 4-ethylphenol, a noneffector of the wild-type protein, was isolated by genetic selection. The single point **mutation** in **DmpR**-E135K, which results in a Glu-to-Lys change in residue 135, also results in a regulator with enhanced recognition of para-substituted methylphenols. The **DmpR**-E135K **mutation**, when introduced into the wild-type strain, confers enhanced utilization of the para-substituted methylphenols. These experiments demonstrate that the aromatic effector activation of wild-type **DmpR** by the para-substituted methylphenols is a major factor limiting the catabolism of these compounds.

L3 ANSWER 23 OF 23 MEDLINE  
ACCESSION NUMBER: 93194783 MEDLINE  
DOCUMENT NUMBER: 93194783  
TITLE: Cloning and nucleotide sequence of the gene encoding the positive regulator (**DmpR**) of the **phenol** catabolic pathway encoded by pVI150 and identification of **DmpR** as a member of the NtrC family of transcriptional activators.  
AUTHOR: Shingler V; Bartilson M; Moore T  
CORPORATE SOURCE: Department of Cell and Molecular Biology, University of Umea, Sweden..  
SOURCE: JOURNAL OF BACTERIOLOGY, (1993 Mar) 175 (6) 1596-604.  
Journal code: HH3. ISSN: 0021-9193.  
PUB. COUNTRY: United States  
Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
OTHER SOURCE: GENBANK-X68033  
ENTRY MONTH: 199306  
AB The catabolic plasmid pVI150 of *Pseudomonas* sp. strain CF600 encodes all the genetic information required for the regulated metabolism of **phenol** and some of its methyl-substituted derivatives. The structural dmp genes of the pathway are clustered in a single operon that lies just downstream of a -24 TGTC, -12 TTGC nif/ntr-like promoter sequence. Promoters of this class are recognized by a minor form of RNA polymerase utilizing sigma 54 (NtrA, RpoN). Primer extension analysis demonstrated that the dmp operon transcript initiates downstream of the -24, -12 promoter. Transposon insertion **mutants**, specifically defective in the regulation of the dmp operon, were isolated, and complementation of a **phenol**-utilization regulatory **mutant** was used to identify the regulatory locus, **dmpR**. The 67-kDa **dmpR** gene product alone was shown to be sufficient for activation of transcription from the dmp operon promoter. Nucleotide sequence determination revealed that **DmpR** belongs to the NtrC family of transcriptional activators that regulate transcription from -24, -12 promoters. The deduced amino acid sequence of **DmpR** has high homology (40 to 67% identity) with the central and carboxy-terminal regions of these activators, which are believed to be involved in the interaction with the sigma 54 RNA polymerase and in DNA binding, respectively. The amino-terminal region of **DmpR** was found to share 64% identity with the amino-terminal region of **XylR**, which is also a member of this family of activators. This region has been implicated in effector recognition of aromatic compounds that is required for the regulatory activity of **XylR**.

L4 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2001 ACS  
ACCESSION NUMBER: 1998:162856 CAPLUS  
DOCUMENT NUMBER: 128:267806  
TITLE: Development and testing of a bacterial  
biosensor for toluene-based environmental  
contaminants  
AUTHOR(S): Willardson, Barry M.; Wilkins, Jon F.; Rand, Timothy  
A.; Schupp, James M.; Hill, Karen K.; Keim, Paul;  
Jackson, Paul J.  
CORPORATE SOURCE: Department of Chemistry and Biochemistry, Brigham  
Young University, Provo, UT, 84602, USA  
SOURCE: Appl. Environ. Microbiol. (1998), 64(3), 1006-1012  
CODEN: AEMIDF; ISSN: 0099-2240  
PUBLISHER: American Society for Microbiology  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB A bacterial biosensor for benzene, toluene, and similar compds.  
was constructed, characterized, and field tested on contaminated H<sub>2</sub>O and  
soil. The biosensor is based on a plasmid incorporating the  
transcriptional activator **xylR** from the TOL plasmid of  
*Pseudomonas putida* mt-2. The **XylR** protein binds a subset of  
toluene-like compds. and activates transcription at its promoter, Pu. A  
reporter plasmid was constructed by placing the luc gene for firefly  
luciferase under the control of **XylR** and Pu. When *Escherichia*  
*coli* cells were transformed with this plasmid vector, luminescence from  
the cells was induced in the presence of benzene, toluene, xylenes, and  
similar mols. Accurate concn. dependencies of luminescence were obtained  
and exhibited K1/2 values ranging from 39.0 .+- . 3.8 .mu.M for 3-xylene to  
2,690 .+- . 160 .mu.M for 3-methylbenzyl alc. (means .+- . std. deviations).  
The luminescence response was specific for only toluene-like mols. that  
bind to and activate **XylR**. The biosensor cells were  
field tested on deep aquifer H<sub>2</sub>O, for which contaminant levels were known,  
and were able to accurately detect toluene deriv. contamination in this  
H<sub>2</sub>O. The biosensor cells also detect BETX (benzene, toluene,  
and xylene) contamination in soil samples. These results demonstrate the  
capability of such a bacterial biosensor to accurately measure  
environmental contaminants and suggest a potential for its inexpensive  
application in field-ready assays.

L4 ANSWER 2 OF 6 DGENE COPYRIGHT 2001 DERWENT INFORMATION LTD  
ACCESSION NUMBER: 1998N-V11356 DNA DGENE  
TITLE: Genetic construct for biosensor - comprises  
reporter gene and gene encoding enzyme, useful for, e.g.  
detection of environmental pollutants  
INVENTOR: Jury K; Schneider R; Vancov T  
PATENT ASSIGNEE: (CRCW-N)CRC WASTE MANAGEMENT & POLLUTION CONTROL  
PATENT INFO: WO 9804716 A1 19980205 52p  
APPLICATION INFO: WO 1997-AU473 19970725  
PRIORITY INFO: AU 1996-1280 19960729  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 1998-145262 [13]  
AB V11353-V11370 are PCR primers used in the construction of a genetic  
construct for use in a biosensor. This biosensor is  
composed of a nucleic acid encoding a reporter and a nucleic acid  
encoding an enzyme which are controlled by inducible promoters. The  
biosensor can measure an environmental signal, e.g. pollutants,  
toxins, temperature, irradiation, biological or chemical signals, and  
also by appropriate choice of promoter it can be used to detect  
physiological responses, e.g. starvation, toxicity or sporulation

L4 ANSWER 3 OF 6 DGENE COPYRIGHT 2001 DERWENT INFORMATION LTD  
ACCESSION NUMBER: 1998N-V11355 DNA DGENE  
TITLE: Genetic construct for biosensor - comprises  
reporter gene and gene encoding enzyme, useful for, e.g.  
detection of environmental pollutants  
INVENTOR: Jury K; Schneider R; Vancov T  
PATENT ASSIGNEE: (CRCW-N)CRC WASTE MANAGEMENT & POLLUTION CONTROL  
PATENT INFO: WO 9804716 A1 19980205 52p  
APPLICATION INFO: WO 1997-AU473 19970725  
PRIORITY INFO: AU 1996-1280 19960729

DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 1998-145262 [13]  
AB V11353-V11370 are PCR primers used in the construction of a genetic construct for use in a **biosensor**. This **biosensor** is composed of a nucleic acid encoding a reporter and a nucleic acid encoding an enzyme which are controlled by inducible promoters. The **biosensor** can measure an environmental signal, e.g. pollutants, toxins, temperature, irradiation, biological or chemical signals, and also by appropriate choice of promoter it can be used to detect physiological responses, e.g. starvation, toxicity or sporulation

L4 ANSWER 4 OF 6 DGENE COPYRIGHT 2001 DERWENT INFORMATION LTD  
ACCESSION NUMBER: 1998N-V11354 DNA DGENE  
TITLE: Genetic construct for **biosensor** - comprises reporter gene and gene encoding enzyme, useful for, e.g. detection of environmental pollutants  
INVENTOR: Jury K; Schneider R; Vancov T  
PATENT ASSIGNEE: (CRCW-N)CRC WASTE MANAGEMENT & POLLUTION CONTROL  
PATENT INFO: WO 9804716 A1 19980205 52p  
APPLICATION INFO: WO 1997-AU473 19970725  
PRIORITY INFO: AU 1996-1280 19960729  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 1998-145262 [13]  
AB V11353-V11370 are PCR primers used in the construction of a genetic construct for use in a **biosensor**. This **biosensor** is composed of a nucleic acid encoding a reporter and a nucleic acid encoding an enzyme which are controlled by inducible promoters. The **biosensor** can measure an environmental signal, e.g. pollutants, toxins, temperature, irradiation, biological or chemical signals, and also by appropriate choice of promoter it can be used to detect physiological responses, e.g. starvation, toxicity or sporulation

L4 ANSWER 5 OF 6 DGENE COPYRIGHT 2001 DERWENT INFORMATION LTD  
ACCESSION NUMBER: 1998N-V11353 DNA DGENE  
TITLE: Genetic construct for **biosensor** - comprises reporter gene and gene encoding enzyme, useful for, e.g. detection of environmental pollutants  
INVENTOR: Jury K; Schneider R; Vancov T  
PATENT ASSIGNEE: (CRCW-N)CRC WASTE MANAGEMENT & POLLUTION CONTROL  
PATENT INFO: WO 9804716 A1 19980205 52p  
APPLICATION INFO: WO 1997-AU473 19970725  
PRIORITY INFO: AU 1996-1280 19960729  
DOCUMENT TYPE: Patent  
LANGUAGE: English  
OTHER SOURCE: 1998-145262 [13]  
AB V11353-V11370 are PCR primers used in the construction of a genetic construct for use in a **biosensor**. This **biosensor** is composed of a nucleic acid encoding a reporter and a nucleic acid encoding an enzyme which are controlled by inducible promoters. The **biosensor** can measure an environmental signal, e.g. pollutants, toxins, temperature, irradiation, biological or chemical signals, and also by appropriate choice of promoter it can be used to detect physiological responses, e.g. starvation, toxicity or sporulation

L4 ANSWER 6 OF 6 MEDLINE  
ACCESSION NUMBER: 1998162061 MEDLINE  
DOCUMENT NUMBER: 98162061  
TITLE: Development and testing of a bacterial **biosensor** for toluene-based environmental contaminants.  
AUTHOR: Willardson B M; Wilkins J F; Rand T A; Schupp J M; Hill K K; Keim D; Jackson P J  
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AB A bacterial **biosensor** for benzene, toluene, and similar compounds has been constructed, characterized, and field tested on contaminated water and soil. The **biosensor** is based on a plasmid incorporating the transcriptional activator **XylR** from the TOL plasmid of *Pseudomonas putida* mt-2. The **XylR** protein binds a subset of toluene-like compounds and activates transcription at its promoter, Pu. A reporter plasmid was constructed by placing the luc gene

for firefly luciferase under the control of **XylR** and **Pu**. When *Escherichia coli* cells were transformed with this plasmid vector, luminescence from the cells was induced in the presence of benzene, toluene, xylenes, and similar molecules. Accurate concentration dependencies of luminescence were obtained and exhibited K<sub>1/2</sub> values ranging from 39.0 +/- 3.8 microM for 3-xylene to 2,690 +/- 160 microM for 3-methylbenzylalcohol (means +/- standard deviations). The luminescence response was specific for only toluene-like molecules that bind to and activate **XylR**. The **biosensor** cells were field tested on deep aquifer water, for which contaminant levels were known, and were able to accurately detect toluene derivative contamination in this water. The **biosensor** cells were also shown to detect BETX (benzene, toluene, and xylene) contamination in soil samples. These results demonstrate the capability of such a bacterial **biosensor** to accurately measure environmental contaminants and suggest a potential for its inexpensive application in field-ready assays.